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Dissociating hyper and hypoself biases to a core self-representation

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ABSTRACT

Biases to favour self-related information over information related to other people have been demonstrated across a range of both high- and low-level tasks, but it is unclear whether these tasks 'tap' the same types of self representation. Here we assess results from two patients with damage primarily to (i) left ventro-medial prefrontal (vmPFC) cortex and the insula (patient SC), and (ii) temporo-parietal (TP) cortex (patient RR). We report evidence from both low-level perceptual matching tasks and episodic memory showing that SC has a hypoself bias across the tasks. RR in contrast had a hyperself bias confined to perceptual matching. Both patients also showed hypobias effects for reward. We argue that the different brain lesions compromise (i) the use of a core self-representation which modulates both perceptual and memorial levels of processing (the vmPFC), and (ii) attentional responses to social cues (the TP cortex), and, furthermore, these effects can dissociate from those of reward and general effects of brain lesion and/or impaired executive control. We suggest that the vmPFC is critical for access to a core self-representation while TP damage can reduce top-down control of attention to salient stimuli and exaggerates the effects of strong (self-related) attentional signals.

1. Introduction

There is now a large amount of evidence indicating that individuals are biased in responding to information relating to themselves compared with information relating to other people. These bias effect emerge upon a wide range of tasks. For example, participants better remember information when judgements are referred to the self compared to when judgements are made in reference to other people (e.g., Cunningham, Turk, & Macrae, 2008; see Symons & Johnson, 1997, for a review). Individuals tend to rate traits more favourably in relation to themselves compared with others (Klein, Loftus, & Burton, 1989). Responses to images of our own faces are faster than to images of other peoples' faces (Ma & Han, 2010; Sui, Liu, & Han, 2009), and images of our own face are difficult to ignore (e.g., Sui, Zhu, & Han, 2006). Self-biases can also modulate lower level tasks such as perceptual matching. For example, Sui, He, and Humphreys (2012) had participants associate a label with a shape (you-circle, friend-square, stranger-triangle). Subsequently they had to decide whether shape-label pairs were the same initially shown (you-circle, friend-square) or whether they were re-paired (you-square, friend-circle). Matching times were substantially faster and more accurate for self-related stimuli than for stimuli related to other people. This advantage for

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perceptually matching self-related stimuli also interacted with the contrast of the shapes (self-related shapes were less affected by lowering the luminance of the stimuli), consistent with an effect on perceptual processing of the stimuli.

One question that arises from this work is whether these different self-bias effects reflect the same underlying information about the self. It has long been argued that different characteristics of the self can be distinguished. For example, William James (1890) separated between a physical representation of the self, mental representations and spiritual aspects. Georg Northoff (2013) discusses the self as a mental entity, a phenomenal experience, as a pre-reflective, implicit coding of experience, and as a social realised representation. Other researchers distinguish a true from an outer self (e.g., Newman, Bloom, & Knobe, 2014) and between cognitive and affective components of the self (Moran, Macrae, Heatherton, Wyland, & Kelley, 2006). Whether there is some common representation that cuts-across these distinctions is a moot point. At a pragmatic level, however, we can ask whether the self information that is accessed in high-level memory and trait-judgement tasks reflects the same information that mediates lower-level perceptual judgements.

One reason to argue for self-based processing relying on some common representation relates to the neural correlates of self processing. For example, it has been found that tasks as dissimilar as making high-level judgements about the self (e.g., Kelley et al., 2002; Mitchell, Banaji, & Macrae, 2005) and perceptual matching of shapes and labels (Sui, Rotshtein, & Humphreys, 2013) all involve activation of the ventro-medial pre-frontal cortex (vmPFC), and that this activation is distinct from activation in other brain areas [e.g., the precuneus, the posterior superior temporal sulcus (pSTS)], which can be differentiated across the tasks (e.g., Moran et al., 2006; Sui et al., 2013). In interpreting their fMRI on the shape-label perceptual matching task, Sui et al. (2013) argued that greater activation for self stimuli over stimuli related to other people in the vmPFC stemmed from activation of self representations, while the similarly enhanced activation of the pSTS was due to increased attentional sensitivity to self-related stimuli.

In the present study we assessed whether there were common representations for the self in high- and low-level tasks by evaluating the effects of brain lesion on self-related processing. We report data from two patients, one with lesions specifically involving the left vmPFC and a second with lesions affecting left temporo-parietal cortex (TPC). Performance was examined in high-level memory tasks, where patients made self-other judgements about stimuli and were then probed for recall and recognition (see Cunningham et al., 2008; Sui & Humphreys, 2013; Van den Bos, Cunningham, Conway, & Turk, 2010), and in the lower-level shape-label perceptual matching task of Sui et al. (2012). We hypothesized that damage to the vmPFC may selectively disrupt access to core representations of the self, recruited for both high- and low-level tasks. If this is the case, then there may be a reduction in the normal self-modulation of performance for a patient with damage to this region, and this should affect memory and perceptual matching alike. In contrast, we propose that the TPC is involved in modulating the attentional response to self-related stimuli. Here the direction of the effects is difficult to predict. One possibility is that self-biases will be reduced due to problems in tuning neurons to self-related items in the environment. A contrasting view is that a reduction in the ability to tune attention may lead to a greater effect of prepotent responses to the self-related stimuli over stimuli related to other people, since there may be few processes resources available for top-down modulation of a response to the self. In this case, a patient with TPC damage may show stronger self-biases, at least in tasks that are strongly determined by attention to the stimuli. We hypothesized that such effects should emerge on the perceptual matching task, where attention to the shape-label combination is an important determiner of performance (see Sui, Yankouskaya, & Humphreys, 2015, for converging evidence). On the other hand, variations in attention should be less critical in a memory task following referential processing in relation to the self or other people, where a single item is presented on each trial and an explicit judgement must be made in relation to it. Here modulation of memory performance by the self may be relatively normal, even in a patient with TPC damage.

The effects for the self on perceptual matching were contrasted with the effects of perceptual matching with reward-related stimuli. One influential account of why self biases merge is that self-related stimuli are inherently rewarding, and so self-biases reflect biases in perception and memory to reward (Northoff & Hayes, 2011). Sui et al. used the associative matching task but paired shapes with different reward values rather than personal labels (e.g., £8-circle; £2-square; £0.5-triangle). After forming the associations participants had to judge whether shape-value pairings matched the original presentation (£8-circle, £2-square), or whether the shape–value pairs were re-arranged (£8-square, £2-circle). At the end of the experiment participants were also given real rewards for each correct response, with the rewards matching the relative values from the different trials. Similarly to the advantage for self-related stimuli over stimuli related to other people, Sui et al. found an advantage for high reward stimuli over low reward pairs. Also this advantage interacted with the manipulation of the contrast of the shapes (high reward shapes showed weaker effects of low contrast). If the self-bias effect and the reward-bias effect stem from a common underlying factor, then we would expect them to be affected similarly by brain lesions. On the other hand, if the underlying factors differ, then it is possible that the effects with the self and reward may dissociate across patients. For example, patients may show an abnormal self-bias [either larger (hyper) or smaller (hypo) than normal] but normal effects for reward associations. It is also possible that there may be dissociations in which a hyperself bias for one factor is combined with a hypo reward-bias for the other. Such dissociation would provide important evidence that self-bias and reward-bias are not reflecting the same factor.

2. Case reports

SC was a right-handed 64 year old male suffered a stroke in February 2012. Fig. 1A illustrates SC's reconstructed lesion map using his MRI scan (3T). His lesion included the left ventromedial prefrontal cortex and the insula extending into

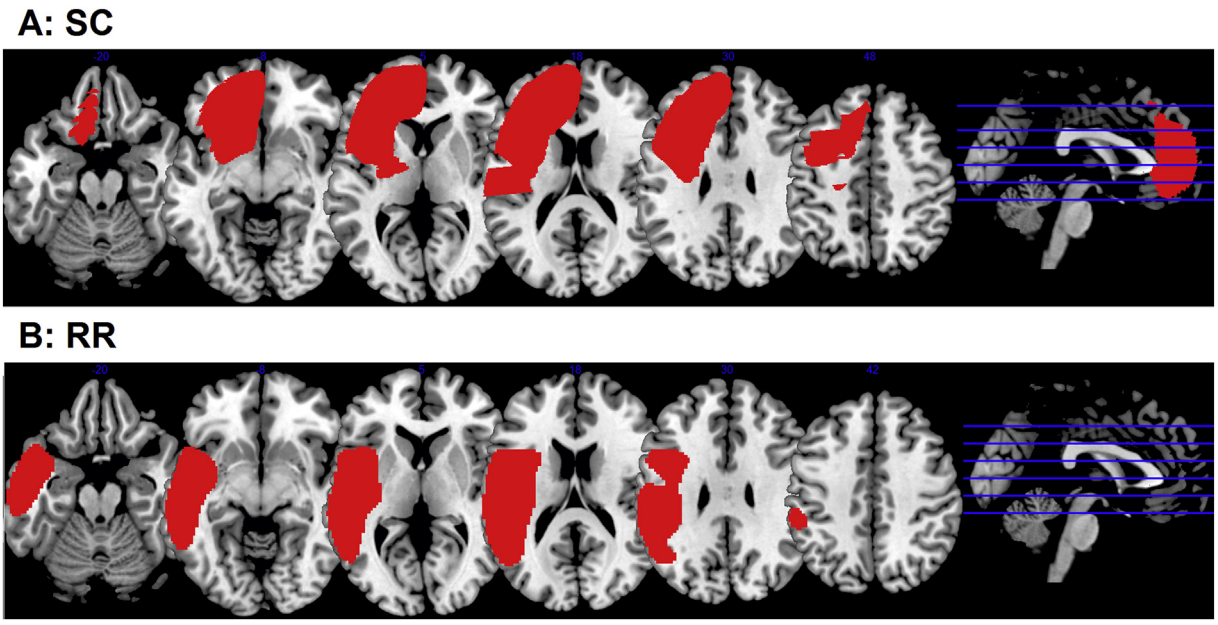

Fig. 1 – (A) ST's lesion and (B) RR's lesions reconstructed using voxel-based morphological analysis relative to control MR scans. Red represents the lesion of grey matter and white matter. The lesion maps were reconstructed by using a modified segmentation procedure and an outlier detection algorithm based on fuzzy clustering (Seghier, Ramlackhansingh, Crinion, Leff, & Price, 2008). This procedure identified voxels that are different in the lesioned brain as compared with a set of 100 healthy controls using normalized gray matter and white matter. The gray matter and white matter outlier voxels are then combined into a single outlier image and threshold to generate a binary map of the lesion. The result of lesion reconstruction was verified against each patient's T1 scan with a threshold of FEW $p = .05$ with ≥100 voxels.

the anterior temporal cortex. SC suffered from executive deficits showing poor performance on the Hayling test (Burgesss & Shallice, 1997) and making multiple rule violations when carrying out the Tower of Hanoi under clinical testing conditions. He also had some word finding difficulties, poor reading, a right-side spatial bias on cancellation and impaired verbal recognition memory. His scores on the Oxford Cognitive Screen (OCS; Demeyere et al., in press) (marked relative to age-matched norms) are shown in Table 1. On clinical tests of social cognition SC had difficulties in performing first-order Theory of Mind tasks (Samson, Apperly, Chiavarino, & Humphreys, 2004).

RR, a former a professional accountant was a right-handed 32 year old male. He suffered a stroke in May 2012. Fig. 1B shows the lesion map reconstructed using RR's MRI scan (3T), with the lesion including the left inferior parietal and superior temporal regions extending into the frontal cortex. The lesion size in these critical regions (the vmPFC and pSTS) is reported in Table 2. RR suffered from severe expressive aphasia, a very poor verbal working memory (matching span 2), some problems in comprehending low imagery words and dyscalculia. His performance on the OCS is also given in Table 1. When initially screened there was impaired naming, reading, writing, verbal recognition memory and executive performance.

Experiment 1. Perceptual matching of self and reward

In the perceptual matching task, we tested whether SC and RR showed normal or abnormal biases in the associative

Table 1 – Summary of performance on the Oxford Cognitive Screen (OCS).

Patient	SC	RR
Sub-test:		
Picture naming ($N = 3$)	**1**	**0**
Semantic recognition ($N = 2$)	2	2
Orientation in time and space ($N = 6$)	6	4
Visual fields ($N = 4$)	4	4
Reading ($N = 15$)	**0**	**0**
Writing numbers ($N = 2$)	2	**0**
Calculations ($N = 4$)	4	4
Hearts cancellation spatial asymmetry (control = 0)	**−8**	0
Imitation ($N = 12$)	11	8
Recognition memory ($N = 4$)	**2**	**0**
Trail making (executive score) (control = 1)	4	**7**

Note. The performance on OCS taken within 7 days on the stroke. The OCS contains 11 sub-tests requiring (i) naming of 3 low frequency pictures (picture naming); (ii) pointing to a named picture amongst semantic distractors (semantic recognition); (iii) forced-choice responses to time and place (orientation in time and space); (iv) visual field confrontation test; (v) reading a passage with regular and irregular words; (vi) writing two numbers; (vii) simple calculations; (viii) cancelling complete hearts and not distractors with a left or right break; (ix) imitating meaningless gestures; (x) recognition memory for words in the reading passage; (xi) the difference between switch and non-switch conditions of a trails test (executive score). In brackets are maximum performance scores. In bold are scores that fall outside the level of age and education matched controls.

Table 2 – The number of lesioned voxels in the vmPFC, left insula, and left posterior parietal-temporal regions. The result of lesion reconstruction was verified against RR and SC's T1 scan with a threshold of FEW $p = .05$ with >100 voxels as compared with a set of 100 healthy controls using normalized gray matter and white matter.

	RR	SC
Medial prefrontal cortex	0	6176
Left angular	2776	0
Left posterior superior temporal region	17,624	2272
Left insula	10,792	12,008

matching procedure of Sui et al. (2012). The procedure required shapes to be associated and then matched with personal labels (you, stranger) or with different reward values (£8 and £0.5). For the reward associations, participants received monetary rewards linked to the associated values for making correct responses to those stimuli in the matching task.

3. Method

Stimuli and tasks. There were two sessions. One involved the personal association task only, the other involved both personal and reward associations. The two sessions took place at least 2 weeks apart. In the *personal association session*, RR and age-matched control participants received three geometric shapes (triangle, circle, square) randomly assigned to labels corresponding to three people (self, friend, or stranger). Associative matching of three shapes and labels was difficult for SC and so for him we required only two associations to be learned; two shapes (triangle and circle) were randomly assigned to the labels for two people (self and friend). Again a similar procedure was followed with age-matched controls.

In the *personal-reward association session* we directly compared associative learning and matching with personal labels and reward values. Four out of six geometric shapes (triangle, circle, square, hexagon, pentagon, and ellipse) were randomly assigned to two personal labels (self and stranger) in the personal associative task (2 shapes), and to the two reward values (£8 and £0.5) in the reward associative task (2 other shapes). The order of the two tasks was counterbalanced across patients and controls.

After the shape-label associative instruction in each task, participants had to carry out a shape-label matching task to discriminate whether pairings of shapes and labels matched, using one of two keys to respond for match and mismatch trials. In the perceptual matching tasks, the shapes were 3.8° × 3.8° in size and appeared above a white central fixation cross (.8° × .8°). A white label stating either 'You', 'Friend', or 'Stranger' (in the personal association task) or '£8' or '£0.5' (in the reward association task) was shown below fixation and covered an area of 1.8°~3.6° × 1.6°. Participants received differential reward values at the end of the reward task reflecting the number of correct responses they made, weighted by the two types of reward association (e.g., an £8 match trial produced 16 times the reward of a £0.5 match trial). All stimuli were displayed on a grey background. E-prime software (version 2.0) was used to present the stimuli and to record the responses. The experiment was run on a PC with a 24-in. monitor and a screen resolution of 1920 × 1080 at 60 Hz.

Procedure. A trial began with a central fixation cross for 500 ms followed by a centrally presented shape-label pair for 100 ms. Participants had to make a judgment whether the pairing of the shape and label matched the original association they had to form, responding within a maximal response time window of 3000 ms, during which a blank screen was presented. They were instructed to make a response by pressing one of two response keys on a keyboard as accurately and quickly as possible. Feedback was given for 500 ms once a response was made and then the next trial started. In each block, the different experimental conditions were randomly presented.

There were twelve or eighteen trials for practice respectively before the two or three association experiment. After practice, participants performed either three blocks of 60 trials for the three associations (patient RR and controls) or three sets of 40 trials for the two association task (patient SC and controls) in the personal associative session. In the personal association–reward association session participants performed one block of 96 trials in each task. Thus, there were 30 trials for each match and mismatch conditions in the personal association session, and 24 trials for each match and mismatch condition in the personal–reward association session. Mismatching trials were defined by the shape that was present. There were 15 trials for each shape re-paired with either one of two mismatched labels (e.g., 15 trials for the self-associated shape re-paired with the 'friend' label and 30 trials for the self-associated shape re-paired with the 'stranger' label) for the 3-pair person; association condition. In the 2-pair association conditions (personal association and reward), the re-paired stimuli were formed by combining each shape with the label for the other shape.

Healthy controls. In order to test the standard self-bias effect, we had 12 age and gender matched healthy controls perform the tasks: 6 matched to patient SC (age: mean ± SD = 63.83 years ± 3.49) (tested on the 2-person associative task), and 6 matched to RR (age: mean ± SD = 27.33 years ± 7.37) (tested on the 3-person association task). A different set of 12 age and gender-matched controls also took part in the personal label-reward session; 6 were matched to patient SC (age: mean ± SD = 59 years ± 10.56), 6 to RR (age: mean ± SD = 25 years ± 6.00).

Data analyses. In order to test the self-bias effects across controls and patients who either carried out two or three personal associations, we only focused on the self-bias indexed by the difference between the self and stranger association, in the personal association session. In the personal–reward association session, we examined the self-bias (self *vs* stranger) and high-reward bias (high *vs* low reward) respectively.

First we assessed the standard self-bias/reward-bias effects from healthy controls. The data on reaction times (RTs) were separately reported for match and mismatch trials due to the different responses being used. Perceptual sensitivity (d prime) was also assessed based on the match and shape-based mismatch trials (see Sui et al., 2012). We assumed that controls would show the advantage in items associated with

the self (and high reward) compared with those associated with stranger (and low reward) condition in both the RTs and d prime. We then conducted comparisons between the performance of the patients and controls using Crawford's modified t-test (for comparing a single case with a group of controls; see Crawford & Garthwaite, 2002; Crawford & Howell, 1998), to examine whether patients showed significant changes in the self-bias and reward-bias effects (either reduced in the patients relative to controls [hypobiases] or increased [hyperbiases]). In order to reduce individual differences in overall reaction times, the data were normalized for each participant before running statistical analyses by calculating the ratio of the difference between the self and stranger (or high and low reward) associations, relative to the sum of the self and stranger (or high and low reward) associations.

4. Results

4.1. *The personal association session*

Controls. For RTs on match trials, a repeated-measures ANOVA with one within-subjects factor – association (self *vs* stranger) and one between-subjects factor – task (three *vs* two personal association) was conducted. The analysis revealed a significant main effect of association, $F(1, 10) = 62.61$, $p < .001$, $\eta^2 = .86$; there were faster responses to the self than to stranger trials. The main effect of task was also significant, $F(1, 10) = 7.94$, $p < .02$, $\eta^2 = .44$, there was better performance for the three than two personal associations. This could reflect differences in the ages of the controls (older for the two association task, to match SC). There was no significant interaction between the association and the task, $F(1, 10) = .39$, $p = .55$ (Fig. 2A). The data replicate the results of prior studies (e.g., Sui et al., 2012) and show that the magnitude of the self bias in normal participants does not change as a function of whether three or two associations had to be formed. We also conducted ANOVAs for the two and three personal association tasks separately. The analysis on the two personal association task showed a significant effect of association, $F(1, 5) = 90.25$, $p = .001$, $\eta^2 = .96$; there were faster responses to the self than to stranger trials. There was also a significant effect of association in the three personal association task, $F(2,10) = 19.42$, $p < .001$, $\eta^2 = .76$; there were faster responses to the self than to friend ($p = .003$) and stranger ($p = .01$) trials, but there was no difference between the friend and stranger trials ($p = .24$).

The analysis for RTs on mismatch trials only showed a significant main effect of task, $F(1, 10) = 7.96$, $p < .02$, $\eta^2 = .44$, which was similar to the results on match trials (RTs for three associations < RTs for two associations). Neither the effect of association nor the interaction between association and task was significant, $F(1, 10) = 3.08$ and 2.65, $p = .11$ and .14.

For d prime, the analysis did not show a significant main effect of association, $F(1, 10) = .24$, $p = .63$ but there was a

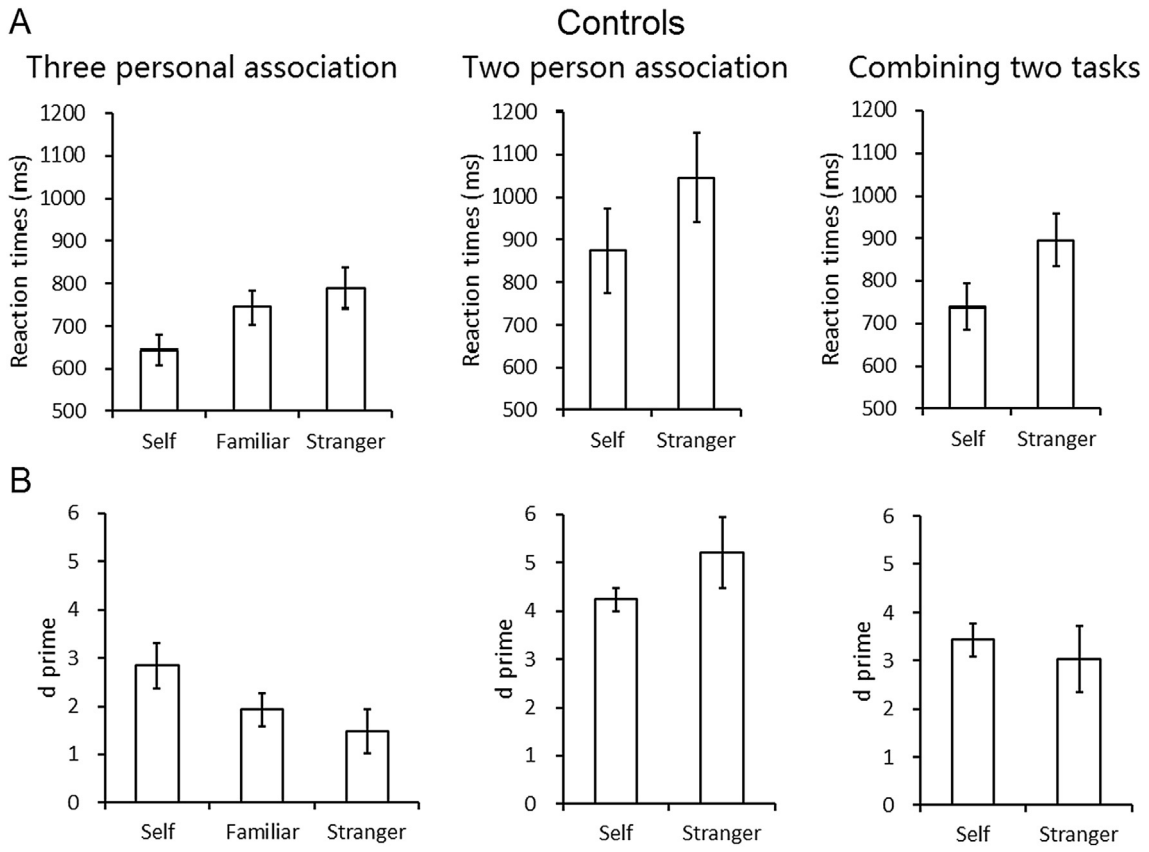

Fig. 2 – Control data in the personal associative session. (A) Reaction times (RTs, ms) as a function of the association (self *vs* stranger). (B) Performance in d prime as a function of the association (self *vs* stranger). Error bars represent one standard error.

significant effect of task, $F(1, 10) = 23.01$, $p = .001$, $\eta^2 = .70$. There was a larger d prime in the two relative to the three personal association conditions. There was also a significant interaction between the association and the task, $F(1, 10) = 7.99$, $p < .02$, $\eta^2 = .48$ (Fig. 2B). For the three personal association task, paired t tests demonstrated larger d prime values for self relative to stranger trials, $t(5) = 4.29$, $p = .005$, but there was comparable performance for self and stranger trials for the two personal association task, $t(5) = -1.21$, $p = .29$. For the d prime analysis, the self advantage emerged for the tasks where more associations had to be formed (three *vs* two associations).

Comparison between patients and controls. We conducted Crawford's modified t-test using normalised scores (see the Method section) to assess whether patients showed a self-bias deficit compared to controls. For RTs on match trials, patient SC showed a significant hypo self-bias deficit relative to the controls performing the two personal associative task, $t(5) = -13.36$, $p < .001$ (Fig. 3A). In contrast, patient RR demonstrated a significant hyper self-bias deficit relative to age-matched controls performing the three personal associative task, $t(5) = 2.72$, $p < .05$ (Fig. 3A).

For d prime, similar to the data on RTs, patient SC showed a significant hypo self-bias deficit relative to controls performing the two personal associative task, $t = -5.51$, $p = .005$ (Fig. 3B). In this case patient RR did not show self-bias deficit relative to controls performing the three personal associative task, $t = -.66$, $p = .54$ (Fig. 3B).

4.2. *The personal–reward association session*

Controls. We first examined the effect of self-bias. Repeated-measure ANOVAs with one within-subjects factor – association (self *vs* stranger) and one between-subjects factor – age (old controls for SC *vs* young controls for RR) were conducted on RTs for match trials, RTs for mismatch trials, and d prime

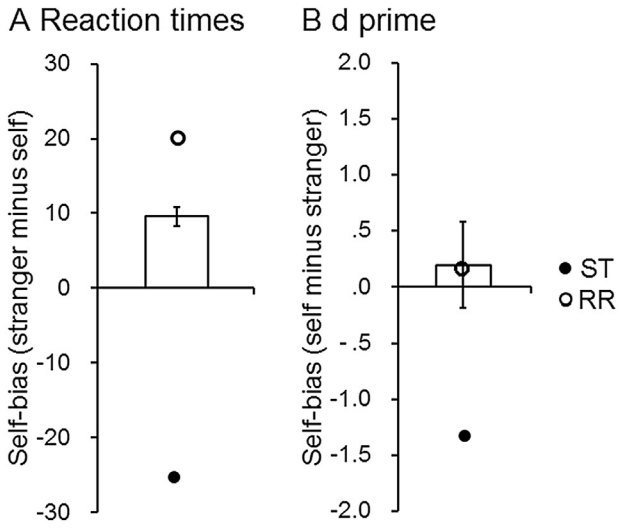

Fig. 3 – The comparison in self-bias between the patients and controls in the personal association session. (A) RTs (ms) and (B) performance in d prime. The bars represent the control data and circles represent each patient's data.

respectively. The analyses did not show any significant main effect of age and no interactions between association and age, $F(1, 10) < 1.38$, $p > .27$. Data from all controls were therefore collapsed. Paired t tests were then conducted for RTs on match trials, mismatch trials, and d prime. The multiple comparisons were corrected by multiple Holm-Bonferroni corrections for $\alpha = .01$ reflecting findings in prior studies (Holm, 1979; Sui et al., 2012). The results showed a significant effect of association on RTs for match trials, $t(11) = -6.24$, $p < .001$; there were faster responses to the self than stranger associations, in line with the results in the personal association session (Fig. 4A). There were comparable responses to the self and stranger associations in RTs for mismatch trials, $t(11) = .92$, $p = .38$. The analysis for d prime failed to show a significant difference between the self and stranger associations, $t(11) = 1.78$, $p = .10$ (Fig. 4B).

For the reward association task (Fig. 4B), there were no significant age effects, $F(1, 10) < 1.37$, $p > .28$. Paired t tests showed a high-reward bias on RTs for match trials, $t(11) = -5.99$, $p < .001$; participants responded faster to items associated with high than low reward values (Fig. 4A). There were no significant effects on RTs for mismatch trials or for the d prime scores, $t(5) = -.13$ and -2.69, $p = .90$ and $.02$ (Fig. 4B).

Comparisons between patients and controls. First we computed the normalized self and reward bias effects. The self/reward-bias in RTs for match trials for each participant was indexed by the difference between the stranger and self conditions (and the low and high reward conditions), dividing by the sum of two responses then multiplying by 100 (Fig. 5A). For SC, Crawford's modified t-test showed a marginally significant hypo self-bias deficit relative to the age and gender matched controls, $t = -2.28$, $p = .04$ (one-tailed). RR demonstrated a significant hyper self-bias deficit relative to age and gender matched controls, $t = 8.07$, $p < .001$ (two-tailed). In the reward association task, both SC and RR showed a significant hypo reward-bias relative to age and gender matched controls, $t = -1.94$ and -3.47, $p = .06$ and $.009$.

For d prime (Fig. 5B), SC showed comparable self-bias effect compared to age and gender matched controls, $t = .06$, $p = .96$. Likewise, RR showed similar effect of self-bias relative to age and gender matched controls, $t = -1.85$, $p = .12$ (Fig. 5B). For the reward-bias, both SC and RR showed comparable performance when compared to age and gender matched controls, $t = .25$ and 1.68, $p = .81$ and $.16$.

5. Discussion

The data indicate that SC and RR had opposite changes in self-bias in perceptual matching after their brain lesions. SC showed a reduced self-bias compared with control participants (a hypo self-bias). RR in contrast had a larger self-bias than that found in controls (hyper self-bias). The results indicate that brain lesions can alter self-bias effects on perceptual matching, though the effects differ according to the lesion site. In addition to this, both patients showed decreased biases to high reward stimuli (hypo reward-bias). The results for RR are particularly striking, since, relative to controls, he had opposite effects on the self- and reward-bias

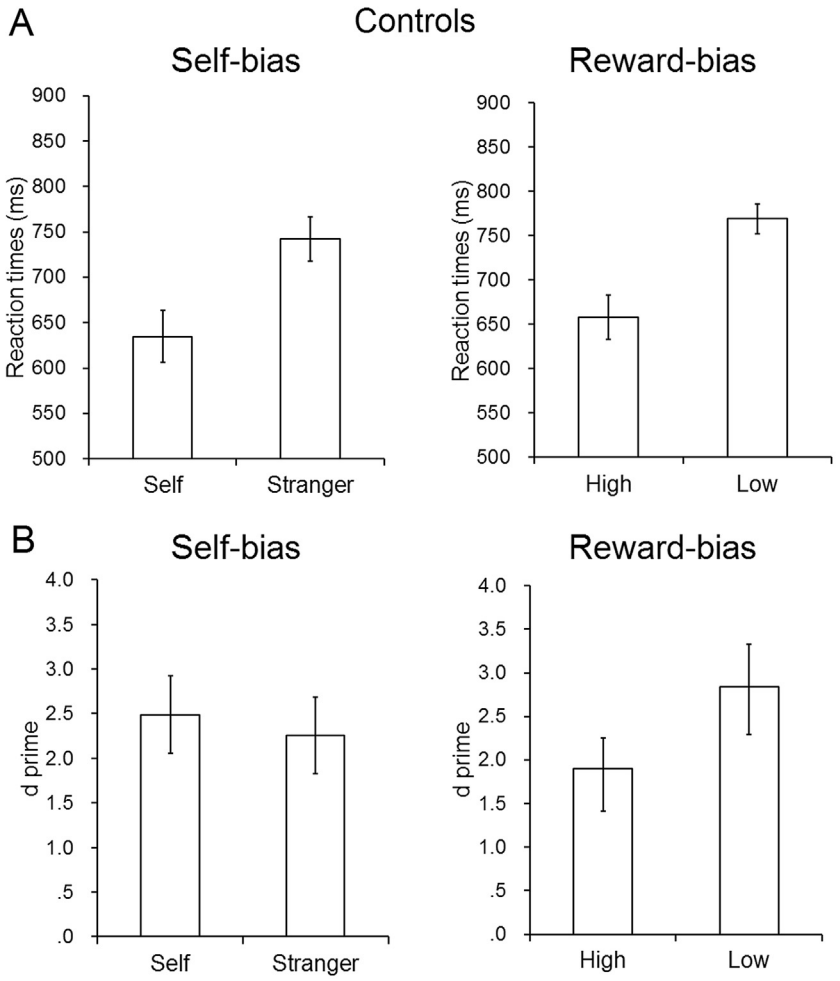

Fig. 4 – Control data in the personal–reward association session. (A) RTs (ms) as a function of the association (self *vs* stranger, or high *vs* low reward). (B) performance in d prime as a function of the association (self *vs* stranger, or high *vs* low reward). Error bars represent one standard error.

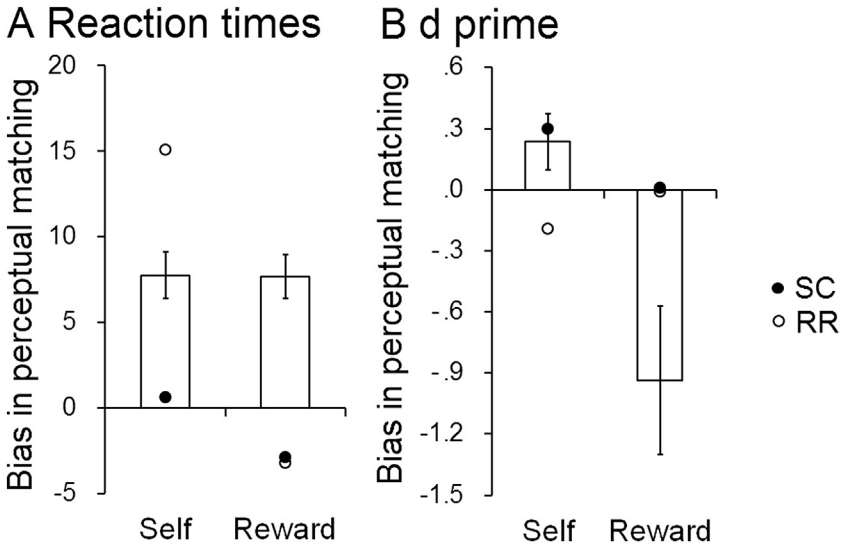

Fig. 5 – The comparison of self/reward-biases between the patients and controls in the perceptual matching tasks. (A) RTs (ms) and (B) performance in d prime. The bars represent the control data and circles represent each patient's data.

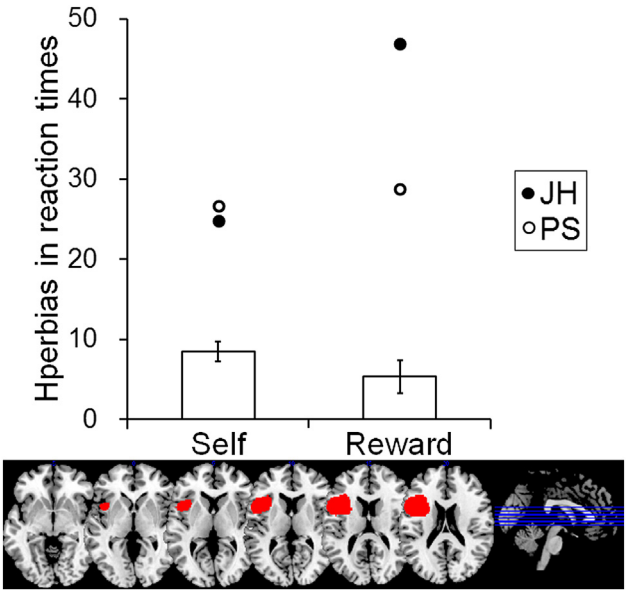

Fig. 6 – Patients JH (53 years old, female) and PS (81 years old, female) show hyperbias for both the self- and high reward-related stimuli. Lesions reconstruction using voxel-based morphological analysis relative to control MR scans illustrates the lesion over the left inferior frontal region extending to the insula.

effects (respectively hyper and hypobias). This contrast supports the argument that the self- and reward-bias effects on perceptual matching can arise from independent sources. We return to this point in the General Discussion.

We note also that these results do not reflect the general effects of brain lesion or the loss of executive control in the patients. First, SC and RR showed opposite effects of self-bias. Second, in similar analyses with patients with lesions affecting the fronto-parietal executive control system we have found that there is a pattern of hyperbias for both self- and reward-related stimuli (see Fig. 6[1]), consistent with a general lack of task based control to salient (self- and high-reward) stimuli along also with difficulty in responding to less strong associations (stranger- and low-reward). The hypodeficits observed in SC (self and reward) and RR (reward) dissociate from the effects seen after fronto-parietal damage.

Experiment 2. Episodic memory

In this experiment, we assessed whether SC and RR not only had changes in self bias in perceptual matching but also in episodic memory. The patients classified objects as related either to themselves or to a familiar other and then we tested recall, recognition of the order of the items and source memory for the location where the objects were positioned.

6. Method

Stimuli and tasks. Participants completed an object naming task as a pre-test before the episodic memory task. Patients were told to name objects taken from the 260 coloured object pictures in Snodgrass and Vanderwart (1980) and after this they evaluated the familiarity of each object based on a 3-point scale (1-do not know, 2-name, not familiar, 3-name, familiar). 120 items which the patients correctly named and marked with 3 were selected and made into cards. 96 items out of 120 were divided into four sub-groups – animals (e.g., ant, butterfly), animate objects (e.g., apple, flower), inanimate indoor objects (e.g., bed, chair), and inanimate outdoor objects (e.g., bike, flag) (see Sui & Humphreys, 2013). These items were classified into 16 subsets each of 6 items.

Each patient underwent three memory tasks, recall items, order and location of presentation, with each subset forming one trial. The location task provided a measure of source memory (where a recalled item had initially appeared). During the encoding phrase, patients were presented with 6 items, one at a time, in a random order. The prior stimulus was left available as new one was presented in a different spatial location. Participants were asked to associate all of the items with themselves (on 'self' trials) or with a familiar other person, chosen by the patients (on 'other' trials). Before each trial, patients were told the type of trial (self *vs* other) and during the encoding stage, they orally reported "this is my chair" or "this is my friend's car". After reporting the ownership for each item, the experimenter put the cards upside down either on the left or right in front of the patients who were asked to remember the location of the items. The items were equally often associated with the self and the other familiar person, and counterbalanced across test sessions. After the items on a trial were presented, the patients had to recall the stimuli. Then the experimenter presented all the items again but in a random order (the recall task). Patients were asked to point the order of items when they were presented earlier (the order task). After this, the patients had to report the location where each item had been positioned. Each of the patients carried out 2 to 4 blocks in each session which took around 45 min. Each block consisted of 4 trials (2 self trials, 2 other trials) and there were 3–4 sessions for each patient (depending upon the testing time available for a session). In total patients performed 10 blocks across 3–4 sessions. The interval between two consecutive sessions was at least two weeks apart. We treated each block as an individual participant to conduct the statistical analyses for each patient (see Sui & Humphreys, 2013).

Healthy controls. In order to test the standard self-bias effect in memory, we had 12 healthy controls carry out the above tasks, 8 matched to patient SC (age: mean ± SD = 61.88 years ± 4.02) and 4 to RR (age: mean ± SD = 29.75 years ± 1.50). Controls were tested in a single session with 3–4 blocks. In the analysis w treated each block per control as a subject, with trials nested within subjects in the analysis.

[1] Fig. 6 shows data from two patients, JH and PS, both of who suffered damage to the left dorso-lateral prefrontal cortex and who presented with deficits in executive functions. For example, both patients scored abnormally on the Hayling test of inhibitory control and both made rule violation errors when carrying out the Tower of Hanoi problem solving task and both were unable to complete the task.

7. Results

Controls. The controls showed better memory for items encoded in relation to the self than for items encoded in related to a familiar other across the three memory tasks, $t(20) = 2.54$, $p = .02$ in the recall task, $t(20) = 2.20$, $p = .055$ in the order task, and $t(20) = 3.12$, $p = .005$ in the source memory task. There was a consistent self-advantage effect across the three memory tasks (Fig. 7A).

Patients. The recall, order judgements and source memory data were analysed using paired *t* tests. The multiple comparisons were corrected by multiple Holm-Bonferroni corrections for $\alpha = .01$ (Holm, 1979; Sui & Humphreys, 2013). SC failed to show any benefit to items encoded in relation to the self compared to items encoded in relation to the familiar other, and this held for the recall, order and source memory tasks, $t(9) = -.29$, $-.45$, and .17, $p = .78$, .66, and .87 (Fig. 7B). One problem here is that SC's performance was at floor in both the recall (just above .20) and source memory tasks (about .50). In order to test whether the lack of any self-advantage for SC was due to his poor performance we tested another patient stroke MM (male, 77 years old) who had an impairment in verbal short-term memory (forward digit span = 2). MM carried out the recall and order tasks similarly to SC but with the items on each trial reduced to 4 (Fig. 7C). With this reduced load, MM's performance was comparable to SC in the recall and order tasks. For the recall task there was a non-significant trend for better performance for the self over other trials, $t(9) = 1.78$, $p = .11$, but MM did show a reliable advantage for items encoded in relation to the self compared with items encoded in relation to the familiar other in the order task, $t(9) = 3.51$, $p = .007$ (MM did not complete the source memory task). The results indicated that the absence of self-bias in memory from SC was not simply due to low overall performance.

In contrast, RR demonstrated a consistent self-bias in the three memory tasks, $t(9) = 2.75$, $p = .02$ in the recall task, $t(9) = 5.67$, $p < .001$ in the order task, and $t(9) = 3.55$, $p = .006$ in the source memory task (Fig. 7D).

Comparisons between patients and controls. In order to assess the memory deficit in patients compared to controls, the self-bias in memory for each participant was normalized in order to reduce individual differences in overall performance. The self-bias was indexed by the difference between the self and the familiar other conditions divided by the sum of the two conditions (Fig. 8). Crawford's modified *t*-test for SC showed a significant hypoself bias deficit in the recall task compared to controls, $t = -2.04$, $p = .03$ (one-tailed), but there was comparable performance between SC and controls in the order and source memory tasks, $t = -.93$ and $-.87$, $p = .38$ and .40. There was also comparable performance between RR and controls across the three tasks, $t = 1.35$, 1.06, and 1.84, $p = .20$, .31, and .09. The data indicated that RR had normal self-bias in memory after brain lesions, whereas the brain lesion in the vmPFC and insula led to a hypoself memory deficit in SC.

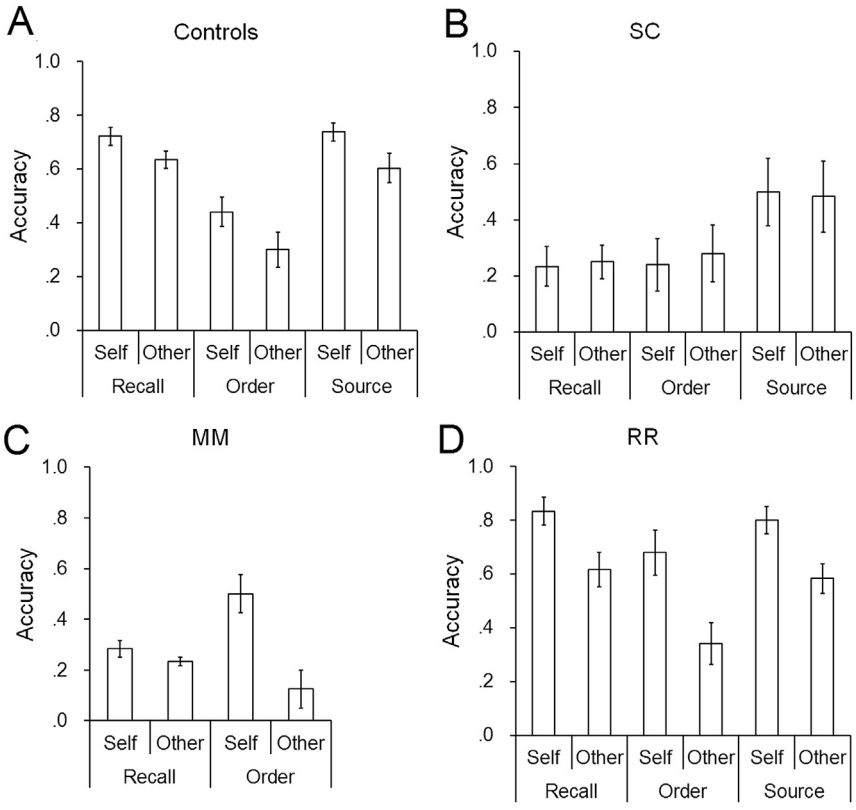

Fig. 7 – Scores for the patients and controls in the recall, order memory, and source memory tasks as a function of the encoding instruction (self *vs* other). (A) controls, (B) SC, (C) MM, and (D) RR. The error bars represent one standard error.

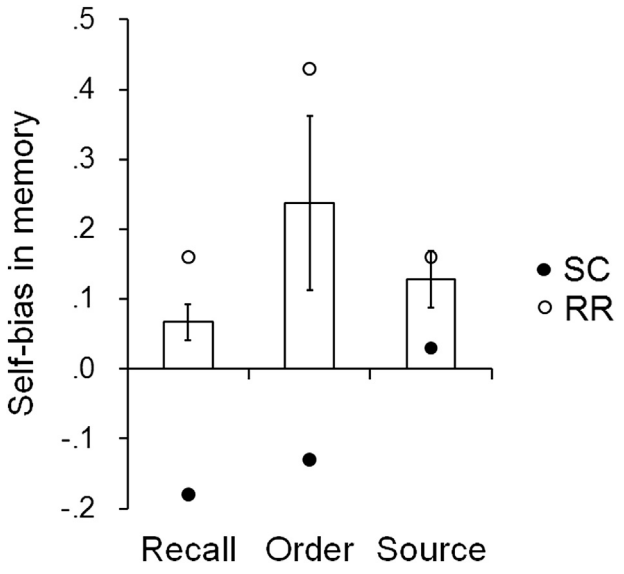

Fig. 8 – Comparisons of the self-bias effect between the patients and controls in the recall, order memory, and source memory tasks. The bars represent the control data and the circles represent each patient's data.

8. Discussion

Patients SC and RR dissociated in the effects of self-coding on episodic memory. For SC there was evidence of a reduced (hypo) self-bias in recall. He also showed no evidence of a self-bias on the order and source memory tasks; though in these last cases the data did not differ from the controls, the controls had reliable self biases in each case. RR, however, demonstrated self-bias effects on the memory tasks that clearly did not differ from the controls. The data indicate that the vmPFC lesion suffered by SC mean that he could not support memory by tagging to a self representation. In contrast, RR could support memory in this way, despite the TP lesions he incurred. Accessing self representations to support memory does not depend on the TP regions lesioned in RR.

9. General discussion

We have reported data on two patients with selective brain lesions affecting areas previously associated with attention to self-related information in perceptual matching (Sui et al., 2013). SC had damage to the vmPFC (plus also the insula). In both perceptual matching and episodic memory, SC consistently failed to manifest self bias effects on his performance; also the differences in responding to self-related stimuli compared to stimuli related to a familiar other were reliably smaller for SC compared with age and education-matched controls. We conclude that SC had a hypo self-bias across both low-level (Experiment 1) and higher-level tasks (Experiment 2). These data contrast with results from RR. RR showed different effects on perceptual matching and episodic memory. For perceptual matching RR demonstrated hyper self-bias effects (greater than those found in age and education matched controls). However for episodic memory RR manifested normal (positive) self bias effects. For RR, then, there was a dissociation across the different levels of task.

To account for these results we suggest that SC's lesion, affecting the vmPFC, disrupted access to a core representations of the self, involved in low- and high-level tasks alike. This argument fits with substantial data from brain imaging in normal participants where the vmPFC is recruited by self-related judgements across a variety of tasks, from making ownership judgements through to matching a shape with a self-related label (Kelley et al., 2002; Mitchell et al., 2005; Sui et al., 2013). In imaging studies, self-biases in perceptual matching but not self-ownership judgements, have also been linked to activity in the posterior superior temporal sulcus (pSTS; Sui et al., 2013) – a region damaged in RR. In interpreting their imaging data, Sui et al. argued that the pSTS mediated top-down attentional responses to socially relevant stimuli, and hence showed a heightened activity in relation to a self-related stimulus. Consistent with this, strong top-down connectivity from the vmPFC to the pSTS predicted the magnitude of the self-advantage in perceptual matching. We propose that damage to the pSTS, in RR, reduces the capacity for top-down attention to be tuned to the stimulus in the perceptual matching task. With the reduction of attentional control, attention is modulated more than usual by the strong social driver of attention – the self-related stimulus: a hyper self-bias results. In contrast, for the episodic memory task there are few demands on attention as the stimuli were sequentially presented and self-bias effects depended more on whether each item could be linked to a core self representation in the vmPFC. Given that RR's lesion falls outside the latter region, this process remains intact (and the magnitude of self-bias effects on memory is normal).

We also presented results on the effects of reward on perceptual matching (Experiment 1). Control participants show positive biases in matching, favouring high-reward stimuli over stimuli associated with lower reward values. The data for both SC and RR indicated hypo reward-biases – that is the patients were less affected by reward than the controls. For SC we might argue that there is a common system for the self and reward that biases processing towards self- and high-reward associated items (see Northoff & Hayes, 2011). However such a common-coding account is contradicted by the results from RR who had opposite biases for the self and reward (respectively hyper and hypo biases). One alternative possibility is that the TP lesion altered the value levels associated with reward, perhaps by disconnecting attentional regions with sub-cortical areas important for coding value (e.g., the ventral striatum). Additional research is required to assess this. The important behavioural point is that the biases favouring the self and high reward can be fractionated in perceptual matching and thus cannot be determined by fully overlapping sources. The hypo reward biases apparent in SC and RR also cannot be attributed to general effects of brain lesions or loss of executive control, as patients with deficits to the fronto-parietal executive network generate hyper biases (Fig. 6). A final caveat is that the results are reported across single cases and we need to exert caution, especially when making inferences about the relations between the brain lesions and the behavioural symptoms – the results should be

replicated in larger group studies. Nevertheless the results do indicate that damage to the vmPFC can disrupt access to a core self-representation mediates both low- and high-level tasks while damage of the left TPC reduces top-down attentional control to self-related stimuli, which particularly affects low-level perceptual matching. These effects on self-bias can dissociate from the effects of reward on performance.

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